THE ROLE OF THE DIAPHRAGM IN THE SENSATION OF HOLDING THE BREATH

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SUMMARY

1. Block of the phrenic nerves in three normal subjects, produced by injection of lignocaine in the neck, caused alleviation of the thoracic sensation during breath holding and prolonged breath-holding time.
2. Injection of lignocaine in the neck without nerve block had no effect on breath holding sensation or breath-holding time.
3. A patient with a spinal-cord transection at the third cervical segment with paralysed diaphragm and chest wall, had no sensation in the chest or abdomen during breath holding.
4. This patient maintained normal ventilation by using hypertrophied sternomastoid muscles. During breath holding he experienced no sensation in the neck despite the presence of sternomastoid contraction.
5. There is previous evidence that complete muscular paralysis abolishes breath-holding sensation but that paralysis of all muscles innervated from spinal segments below the eighth cervical has no effect.

Fowler (1954) showed that the sensation at the breaking point of breath holding is completely relieved by breathing a mixture of 8.2% oxygen and 7.5% carbon dioxide, i.e. the sensation is relieved by the resumption of respiratory movement even though asphyxia is not relieved.

A possible interpretation of this result is that during breath holding a change takes place in the thorax which results in an alteration of afferent nervous information, interpreted by the brain as the unpleasant sensation of breath holding. The sensation is abolished by bilateral local anaesthesia of the vagus nerves which blocks afferent nerve fibres from the lungs (Guz, Noble, Widdicombe, Trenchard, Mushin & Makey, 1966a); it is not known what the important change in the lung is or which lung receptors are involved. Campbell, Freedman, Clark, Robson & Norman (1967) and Campbell, Godfrey, Clark, Freedman & Norman (1969) showed that the sensation was also abolished by complete muscular paralysis with curare. This

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experiment showed that the sensation does not occur if muscles are prevented from contracting.

During breath holding irregular contraction of the inspiratory muscles takes place which increases in strength and frequency as breath holding proceeds. This muscular activity can be demonstrated by electromyography (Agostoni, 1963). It is presumably the abolition of these contractions which is responsible for the effect of muscular paralysis on the breath-holding sensation. Eisele, Trenchard, Burki & Guz (1968) began an analysis to determine which muscles are important in this phenomenon by blocking the nerves to the chest wall of patients undergoing urological surgery. Spinal anaesthesia was raised to include the first thoracic segment, so that the intercostal muscles were paralysed but the diaphragm (supplied by the third, fourth and fifth cervical segments) and accessory muscles of respiration remained intact. This procedure caused no change in the sensation during breath holding and breath-holding time was unaltered. This proved that the intercostal muscles were not involved in the genesis of the sensation when other muscles were able to contract. An additional and incidental conclusion from this experiment was that the sympathetic nervous system, which is also blocked by the procedure of Eisele et al. (1968), is not involved in the genesis of the sensation under these circumstances.

The principal respiratory muscle with intact function during chest wall block is the diaphragm. Eisele et al. (1968) therefore postulated that the sensation during breath holding (the onset of which coincided with the onset of diaphragmatic activity) arose directly from the contractions of this muscle. The present investigation was designed to test this hypothesis. In view of the results of vagal blockage on breath-holding sensation (Guz et al., 1966a), it is also postulated that the diaphragmatic contractions result from an afferent vagal drive which itself is not appreciated by consciousness (Guz et al., 1966a). Observations have been made in a patient with high cervical spinal-cord transection to obtain evidence regarding this suggestion. The observations on breath-holding sensation in this patient and the data in subjects M.N., J.E. and S.B. were discussed at a recent Ciba Foundation Symposium (Guz, Noble, Eisele & Trenchard, 1970; Noble, Eisele, Trenchard & Guz, 1970).

METHODS

Phrenic nerve block

Subjects. Block of the phrenic nerves was attempted by injection of local anaesthetic on both sides of the neck. The subjects were M.N., male, aged 32 (a physician), J.E., male, aged 35 (an anaesthetist), S.B., male, aged 19, N.V., male, aged 22, N.N., female, aged 26, J.W., male, aged 26, J.C., male, aged 21, B.C., male, aged 23, D.C., male, aged 25 and A.C., male, aged 30. All were normal volunteers. M.N. and J.E. were acquainted with the research. The remainder were unacquainted with physiology.

Method of nerve block. We used a percutaneous method of lignocaine block as described by Sarnoff & Sarnoff (1951). A needle, which was insulated electrically except at its tip, was introduced at the phrenic nerve point (Duchenne de Boulogne, 1873) behind the lower end of the sternomastoid and anterior to the scalenus anterior muscle. Electrical stimuli were passed through the tip of the needle. The position of the needle was adjusted until the stimuli produced a powerful diaphragmatic jerk indicating that the needle tip was close to the phrenic nerve. Local anaesthetic (2% lignocaine or 2% lignocaine plus 0.2% tetracaine) was then injected through the needle until the stimuli no longer produced any diaphragmatic contractions.
**Diaphragmatic sensation in breath holding**

_Criterion of successful nerve block._ The success of the block was determined by measuring the change in inspiratory capacity by using a Wedge spirometer with the subject sitting. According to Bergofsky (1964) the descent of the diaphragm contributes 26% of the total tidal volume in the erect posture. Unilateral phrenic nerve crush produces a decrease in vital capacity of 15–18% (Vaccarezza, Soubrie & Rey, 1948; Siebens, Storey, Newman, Frank & Swenson, 1955). Bilateral phrenic block might therefore be expected to produce a 30–36% decrease in vital capacity and approximately the same fall in inspiratory capacity (since the diaphragm does not contribute to expiration). The changes in lung volumes of our subjects are listed (Table 1).

<table>
<thead>
<tr>
<th>Table 1. Subjects studied by lignocaine injection into the neck</th>
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<td>Subject</td>
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<tr>
<td><strong>Experimental group</strong></td>
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<tr>
<td>M.N.</td>
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<td>J.E.</td>
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<td>S.B.</td>
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<td><strong>Control group</strong></td>
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<td>N.V.</td>
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<td>J.W.</td>
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<td>B.C.</td>
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We have taken those cases where the inspiratory capacity was decreased by 20% or more as showing considerable diaphragmatic paralysis although this was not complete as judged by fluoroscopy. In subjects N.V., N.N., J.W., J.C. and B.C., the procedure was unsuccessful, presumably because the needle tip was not close enough to the nerve or because the anaesthetic required for motor block was greater in these individuals.

_Criteria of concomitant vagal block._ Since vagal block abolishes the sensation of breath holding (Guz et al., 1966a) it is necessary to exclude from the study those subjects with any evidence suggestive of vagal block. In all the subjects except J.E. intravenous atropine (1.8 mg) was given before inserting the needle, to protect the subject against possible stimulation of the vagus nerve by the needle tip, which might produce bradycardia or cardiac arrest. In these subjects the heart rate could not be used as evidence for or against vagal block. J.E. (the anaesthetist) deliberately took the risk of not having the efferent vagal fibres blocked with atropine and did not develop tachycardia after lignocaine injection. (In an earlier, unsuccessful attempt, J.E. developed tachycardia and hoarseness.)

In the other subjects the criteria of change in voice and of change in ventilatory responsiveness to carbon dioxide (CO₂) were used. Hoarseness after lignocaine injection in the neck may be due to involvement of those fibres in the main trunk of the vagus which are going to the recurrent laryngeal nerve. Guz, Noble, Widdicombe, Trenchard & Mushin (1966b) and Guz et al. (1970) using the rebreathing method of Read (1967) have shown that vagal block produces a decrease in the ventilatory response to CO₂ and, in particular, a failure of the respiratory rate to rise with increasing PCO₂. This is also true in other species (Scott, 1908; Sasaki, 1927; Florez & Borison, 1967; Richardson & Widdicombe, 1969; Guz et al., 1970).
J.C. became hoarse, N.N. had a lowered increase in respiratory rate in response to CO₂, A.C. showed a dramatic decrease in CO₂ responsiveness and D.C. showed a smaller but statistically significant decrease in the slope of the CO₂ response curve. The data from these subjects, who all had prolongation of breath holding time after lignocaine injection, have therefore been rejected.

The remaining subjects were divided into two groups. (a) ‘Experimental’ consisting of those subjects with phrenic block but no vagal block, i.e. M.N., J.E. and S.B. (b) ‘Control’ consisting of those subjects with neither phrenic block nor vagal block but with similar amounts of lignocaine injected into the neck, i.e. N.V., J.W. and B.C.

Cervical spinal-cord transection

Patient R.A., male aged 24, was studied on several occasions during the third year after a fracture dislocation of the second and third cervical vertebrae. The patient was maintained on positive pressure respiration for 1 year. For 1 year before commencing the studies he had been breathing spontaneously by using accessory muscles of respiration, particularly the sternomastoids which were greatly hypertrophied. His vital capacity was 860 ml and end-tidal $P_{CO_2}$ 35 mmHg. Clinical examination at the time of study revealed a complete motor lesion below the third cervical segment (C3). The sensory lesion was also complete below C3 to objective clinical testing, but the patient was aware of feeling something in small areas in the arms and abdomen. The evidence that his chest wall and abdomen were completely paralysed was the lack of movement of these parts as judged by clinical observation, cinephotography, fluoroscopy and cinefluoroscopy. Additional evidence of diaphragmatic paralysis was obtained by recording the diaphragmatic electromyogram (Fig. 1) with an oesophageal electrode positioned at the dia-
phragm under radiographic control. A tracheostomy had been performed on this patient at the time of admission and was still present at the time of study.

No lignocaine injections were made in this patient. The integrity of afferent vagal innervation was tested by recording the patient’s observations concerning the sensation produced by the insertion of a catheter for suction of bronchial mucous. He described a sense of irritation and desire to cough, often accompanied by lacrimation (Noble et al., 1970).

Protocols

The standard procedure was to allow the subject to breath for 5 min wearing a polythene face mask through which 100% oxygen was administered. This was done to avoid the development of hypoxia during subsequent breath-holding manoeuvres. End-tidal $PCO_2$ was measured by using a Beckman LB-1 analyser. The subject was asked to hold his breath at the end of a normal breath out, i.e. at functional residual capacity (FRC). The duration of breath holding was recorded with a stop watch. When normal breathing recommenced, the subject was asked to describe the sensation he had experienced while breath holding which had finally forced him to take a breath.

In the phrenic block experiments this procedure was carried out before and after the injection of lignocaine in the neck in both control and experimental groups. The procedure was different in J.E. in that he held his breath after breathing air before and after the injection of lignocaine.

The standard procedure was also carried out in the patient with cervical spinal cord transection. In addition, a right sternomastoid electromyogram was recorded with a surface electrode, Medelec MS/3 amplifier and C6/4 oscilloscope, the screen of which was photographed with a Cossor camera. The patient (with tracheostomy tube) was unable to close his glottis to prevent movements of air resulting from spontaneous involuntary muscular contraction. The tracheostomy tube was therefore made airtight and occluded during breath holding. The occlusion was relaxed when the patient signalled that he wanted to breathe. Diaphragmatic contractions were recorded with an abdominal pneumogram.

RESULTS

There was no change in the breath-holding sensation of the control subjects (N.V., J.W. and B.C.) after injection of lignocaine into the neck. M.N. described his sensations during phrenic block thus, ‘I felt the usual sensation come on at the usual time but with reduced intensity. It is my usual practice when breath holding in the control state to resist this sensation until the last possible instant by twisting my body and limbs. I therefore resisted in the same way during phrenic block and found that the sensation disappeared for some time and then came back toward the break point. The break point was similar to the control’. J.E. said (during phrenic block) ‘The sensation was similar in nature but greatly reduced in intensity’. S.B. said (during phrenic block) ‘The sensation during breath holding was felt in a higher area in the chest’.

The breath holding times (BHTs) of these subjects are shown in Fig. 2. Injection of lignocaine had no effect on BHT in the control subjects (N.V., J.W. and B.C.) but markedly increased BHT in the experimental subjects (M.N., J.E. and S.B.). In each case the end-tidal $PCO_2$ at the start of breath holding was similar during the control period and during the block. The increased BHT during phrenic block was therefore not due to hyperventilation before breath holding, which would have lowered the end-tidal $PCO_2$. 

R.A., the patient with C3 transection, felt nothing in his chest during breath holding. He held his breath until 'things started to go black'. The sensation which made him break was in the head and was giddiness. His breath-holding time was 120 s. During breath holding slow rhythmical bursts of electrical activity were recorded from the right sternomastoid muscle (Fig. 3). No arterial blood samples were available for blood gas analysis in this patient.

**DISCUSSION**

These experiments together with those of Eisele et al. (1968) suggest that the diaphragm is involved in the sensation of breath holding.

The first approach described here, namely phrenic block, had the advantage that no muscles other than the diaphragm were paralysed, the BHT could be measured before and after the block and the results could be compared with those in control subjects who had similar amounts of lignocaine injected into the neck without producing block. In all three phrenic block subjects the sensation was decreased or altered and BHT prolonged whereas there was no effect in the control subjects. This shows that the diaphragm participated in the genesis of the breath-holding sensation but does not exclude a contribution from other muscles. Complete abolition of the sensation would not be expected because the block was not complete. A change in functional residual capacity can produce a change in BHT. However, it would be necessary to breathe in to total lung capacity to produce doubling of BHT such as accompanies phrenic block (Fig. 2).
Diaphragmatic sensation in breath holding

All muscles innervated from spinal segments T1 and below have already been excluded from a part in the genesis of the sensation of breath holding when muscles above T1 are innervated (Eisele et al., 1968). However, it is possible that accessory muscles of respiration innervated by segments above T1 also contributed to the sensation.

It is for this reason that the study in R.A., the patient with high cervical cord transection (C3), is of great importance. He breathed entirely with his accessory muscles of respiration innervated by the XIth cranial nerve and cervical segments above C3. If these muscles contributed to the sensation during breath holding, one would not expect complete abolition of this sensation in such a patient. In fact, it was completely abolished, in spite of the fact that vigorous sternomastoid contraction took place (Fig. 3). The patient felt nothing in the chest, neck or throat. The results in this patient show that muscles innervated from spinal segments above C3 are unlikely to be involved in the breath-holding sensation. There appears to be an important

![Fig. 3. Right sternomastoid EMG in patient R.A. during breath holding (between the arrows). Continuous record showing a build-up of electrical activity in this muscle as breath-holding proceeds.](image)

contribution therefore from muscles innervated by spinal segments above T1 (Eisele et al., 1968) and below C3 (patient R.A.). The principle muscle of respiration innervated by segments C3–C8 is the diaphragm. The BHT of patient R.A. is of little value in the absence of a control value. His BHT and those of the subjects with phrenic nerve block are within the very wide range of values found in normal intact subjects.

If the unpleasant sensation during breath holding arises from diaphragmatic contractions, one might expect a similar sensation during phrenic nerve stimulation. This was experienced by the normal subjects during the procedure for localizing the nerve. This was an unpleasant sensation but differed from that during breath holding in the same way that the contractions themselves differed, a short sharp contraction feeling like a hiccup after each electrical stimulus and a slow build-up of sensation during breath holding consistent with the slow build-up of diaphragmatic activity (Agostoni, 1963).

We have no information as to which receptors in the diaphragm are involved in the final peripheral afferent pathway of this sensation. The muscle spindles are obvious candidates, but are much less numerous in the diaphragm than in the intercostal muscles (Dogiel, 1902; Gregor, 1904; Masumoto, 1934a, b; Winckler & Delaloye, 1957) which are rich in spindles.
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(Kreschner, 1888; Huber, 1902), but do not take part in the genesis of the breath-holding sensation. There are about as many tendon receptors as muscle spindles in the diaphragm (Yasargil, 1962; Corda, von Euler & Lennерstrand, 1965). Being in series with the contractile part of the diaphragm, these tendon receptors are better suited to the role of 'tension receptors'. Small muscle afferent fibres in the phrenic nerves (Landau, Akert & Roberts, 1962) could also be involved.

The patient with C3 transection is also of interest in that the rhythmic contractions of the sternomastoids during breath holding presumably result from a central inspiratory drive. The contractions (Fig. 3) are similar to those which occur in the diaphragm of normal subjects during breath holding (Agostoni, 1963). The drive to the diaphragm is thought to be due to a reflex with an afferent vagal pathway (Noble et al., 1970). We do not know whether the drive to the sternomastoids also depends on the integrity of vagal afferent fibres since we have not done a vagal block in this patient. However, whatever the mechanism which produced this drive, it is remarkable that such a drive should be present and voluntarily suppressed, and yet should not be felt as a sensation.

REFERENCES


Diaphragmatic sensation in breath holding


